Jugular venous desaturation and outcome after head injury

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Abstract

Early experience with continuous monitoring of jugular venous oxygen saturation (SjvO₂) suggested that this technology might allow early identification of global cerebral ischaemia in patients with severe head injury. The purpose of the present study was to examine the relationship between episodes of jugular venous desaturation and neurological outcome. One hundred and sixteen severely head-injured patients had continuous monitoring of SjvO₂ during days 1–5 after injury. Episodes of jugular venous desaturation (SjvO₂ < 50% for more than 10 minutes) were prospectively identified, and the incidence of desaturation was correlated with neurological outcome: 77 episodes of desaturation occurred in 46 of the 116 patients; 27 had one episode and 19 had multiple episodes of desaturation. The causes of these episodes were systemic ($n = 36$), cerebral ($n = 35$), or both ($n = 6$). Most of the episodes were less than 1 hour in duration, and it is probable that many of them would not have been detected without continuous measurement of SjvO₂. Episodes of desaturation were most common on day 1 after injury, and were twice as common in patients with a reduced cerebral blood flow as in patients with a normal or elevated cerebral blood flow. The occurrence of jugular venous desaturation was strongly associated with a poor neurological outcome. The percentage of patients with a poor neurological outcome was 90% with multiple episodes of desaturation and 74% in patients with one desaturation, compared to 55% in patients with no episodes of desaturation. When adjusted for all co-variates that were found to be significant, including age, Glasgow coma score, pupillary reactivity, type of injury, lowest recorded cerebral perfusion pressure, and highest recorded temperature, the incidence of desaturation remained significantly associated with a poor outcome. Although a cause and effect relationship with outcome cannot be established in this study, the data suggest that monitoring SjvO₂ might allow early identification and therefore treatment of many types of secondary injury to the brain.

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A number of physiological factors have been related to outcome after severe head injury. Intracranial hypertension, systemic hypotension, arterial hypoxia, and hypocapnia have been implicated as causes of cerebral hypoxia/ischaemia and have been associated with a poor neurological outcome.[1-6]

Early experience with continuous monitoring of jugular venous oxygen saturation (SjvO₂) using a fibroptic catheter had suggested that this technology might allow early identification of both cerebral ischaemia resulting from systemic and cerebral causes and transient episodes of ischaemia which would otherwise go undetected and untreated.[7-12] More recent studies have suggested that SjvO₂ monitoring might help determine optimal hyperventilation[13] and, combined with transcranial Doppler measurements, optimal cerebral perfusion pressure.[14 15] The purpose of the present study was to examine the relationship between episodes of jugular venous desaturation and neurological outcome in patients who had sustained a severe head injury.

Patients and methods

PATIENT POPULATION AND MANAGEMENT

Between 15 July 1989 and 30 September 1992, 116 patients admitted consecutively to Ben Taub General Hospital, Houston, Texas, USA, with a severe head injury (Glasgow coma scale, GCS ≤ 8) had continuous measurements of SjvO₂ using a fibroptic oxygen saturation catheter (4 French gauge catheter, Abbott Laboratories, North Chicago IL, United States) positioned in the jugular bulb. The accuracy of the oxygen saturation measurements obtained with this catheter has been described in a previous publication.[12] The protocol was approved by the Baylor Institutional Review Board and informed consent was obtained from the families of the patients participating in the study.

Ninety per cent of the patients studied were male; most were between the ages of 16 and 35, with an average age of 31·5 (SD 14·7) years. Eighty-six per cent of the patients studied had a closed head injury, whereas the remaining 14% had a gunshot wound to the head. Eighty per cent of the patients were comatose on admission to the hospital. The GCS after resuscitation was 3–5 in 34% of the patients and 6–8 in 46%. Twenty per cent of the patients had an initial GCS of >8, but deteriorated to coma within 48 hours of injury. The average GCS was 6·9 (SD 3·0) on

arrival in the emergency room, and 6·8 (SD 2·3) on day 1 after injury.

All patients were managed with a protocol that emphasised prompt evacuation of intracranial haematomas and prevention of secondary insults to the brain. Patients were intubated and ventilated to maintain a PaO_2 of at least 100 mmHg and a $PaCO_2$ of about 35 mmHg. Intracranial pressure (ICP) was monitored, usually by ventriculostomy, and pressure >20 mmHg were treated. Intracranial hypertension was treated with cerebrospinal fluid drainage, hyperventilation ($PaCO_2$ 25–30 mmHg), sedation, paralysis, and mannitol. Mean arterial blood pressure (MAP) was continuously monitored and maintained so that cerebral perfusion pressure (CPP) was at least 60 mmHg. Barbiturate coma was used only if intracranial hypertension was refractory to the above regimen. Other routine medications included phenytoin and antibiotic drugs.

CONTINUOUS PHYSIOLOGICAL MEASUREMENTS
To monitor $SjvO_2$, the fibroptic catheter was inserted into the internal jugular vein through a 4·5 French gauge peel-away introducer (Cook Critical Care, Bloomington Ind., United States), and threaded into the jugular bulb. The catheter for measurement of $SjvO_2$ was placed on the right side unless the left jugular venous circulation was demonstrated to be dominant: 96 (83%) were placed on the right side and 20 (17%) on the left. A radiograph was obtained to ensure that the catheter was correctly positioned in the jugular bulb. Every effort was made to start the monitoring as early after admission as possible. The catheter for measurement of $SjvO_2$ was inserted an average of 16·6 (SD 15·0) hours after admission to the neuro-intensive care unit: 93 (80%) were inserted within 24 hours of admission. Where monitoring was started more than 24 hours later, reasons included: inability to obtain informed consent from families (n = 6), early contraindication for jugular catheter such as coagulopathy or suspicion of a cervical spine injury (n = 3), and neurological deterioration after 24 hours (n = 14). Patients were monitored for an average of 88·2 (SD 75·5) hours.

The following physiological parameters were recorded at an interval of 1–2 minutes, stored in a computerized database, plotted against time, and correlated with clinical events and intermittent measurements of cerebral blood flow (CBF) and cerebral metabolism: ICP, mean arterial blood pressure (MAP) via a radial artery cannula, CPP, arterial oxygen saturation (SaO_2), end-tidal CO_2, and $SjvO_2$. The catheter for measurement of $SjvO_2$ was calibrated before insertion using the standard provided by the manufacturer. In addition, an in vivo calibration was performed after insertion, and every eight hours thereafter, if the difference between the catheter value and the SO_2 measured in a blood sample drawn through the catheter was more than 4%.

IDENTIFICATION AND MANAGEMENT OF JUGULAR VENOUS DESATURATION
Episodes of jugular venous desaturation, defined as an $SjvO_2$ <50% for more than 10 minutes, were identified prospectively. The first task was to verify the accuracy of the catheter at the time of the desaturation, this is because, as discussed in a previous report, the catheter is subject to artefactual readings from movement of the catheter within the jugular bulb.[12] In the 116 patients, 172 episodes of jugular venous desaturation were identified initially. On investigation, 95 (55·2%) of the episodes could not be confirmed by measuring SO_2 in a blood sample drawn through the catheter.

Once it was confirmed that the $SjvO_2$ was <50% in a blood sample drawn through the catheter, it was necessary to identify the cause. Systemic causes of reduced oxygen delivery were sought by examining blood pressure, arterial blood gases, and haemoglobin concentration; ICP and CPP were noted. If an obvious cause was not found, cerebral blood flow was measured. When a remediable cause for the desaturation was found the appropriate treatment was applied.

INTERMITTENT MEASUREMENTS OF CBF AND METABOLISM
CBF was measured by the nitrous oxide saturation method every eight hours.[16] A total of 1042 measurements of CBF or an average of nine measurements per patient (range 1–29 per patient) were obtained. Arterial and jugular venous blood samples were obtained together with measurements of CBF for determination of PaO_2, $PaCO_2$ pH, oxygen saturation, lactate, and haemoglobin concentration. A blood gas analyser was used to measure PaO_2, $PaCO_2$, and pH. Oxygen saturation and haemoglobin concentration were measured on a co-oximeter (IL-282, Instrumentation Laboratories, Lexington Mass., United States). Patients were classified as having a normal, elevated, or reduced CBF, through comparison of the individual measurements with the normal range for CBF at the patient's $PaCO_2$, as described in a previous publication.[16]

DETERMINATION OF NEUROLOGICAL OUTCOME
Outcome was determined three months after injury by personnel who were unaware of the desaturation information. The Glasgow outcome scale (GOS) was used to quantify the severity of the neurological deficits. For comparison of the outcome with the $SjvO_2$ data, a favourable outcome was defined as a GOS score of good recovery or moderate disability, and a poor outcome was defined as a GOS score of severe disability, vegetative, or dead.

STATISTICAL METHODS
Summary data are reported as the mean ± standard deviation. Categorical variables were analysed using χ-square whereas differences in means were tested using a one-way analysis

of variance. Statistical significance was defined as p < 0·05 for primary effects.

Multiple logistic regression analysis was used to determine the contribution of more than one variable to the outcome and to the occurrence of desaturations. The model was fitted in a forward selection manner. Covariates in the multiple logistic regressions were included when p < 0·20. The final model contains only those additional variables that were significantly associated with outcome.

Results

CONFIRMED EPISODES OF JUGULAR VENOUS DESATURATION

Seventy-seven episodes of jugular venous desaturation, defined as $SjvO_2$ <50% for more than 10 minutes, were identified prospectively and confirmed in 46 of 116 patients. The episodes were confirmed by measuring SO_2 in a blood sample drawn through the catheter.

The $SjvO_2$ values at the time of the identification ranged from 17% to 49%, averaging 39·3 ± 11·7%.

Twenty-seven (23·3%) of the patients had one episode, 11 (9·5%) two episodes, and eight (6·9%) more than two episodes of desaturation. Demographic characteristics, type of injury, severity of injury, and presence of systemic injuries were not significantly related to the incidence of desaturation (table 1).

The incidence of jugular venous desaturation was more common early after injury. Twenty-eight (36·8%) of the 77 episodes of desaturation occurred within 24 hours of admission to the neuro-intensive care unit. There were 13, 10, 12, and seven episodes on days 2–5 after injury, respectively. Only seven episodes occurred after day 5, with a mean onset of the episodes of desaturation of 53·9 (SD 47·9) hours after admission to the non-intensive care unit.

Duration of the 77 episodes of desaturation ranged from 15 minutes to five hours; 57 (75%) were less than one hour. The mean duration of all the episodes was 1·2 (SD 1·6) hours.

The causes of the jugular venous oxygen desaturation could be identified as having a cerebral origin in 35 episodes, arising from intracranial hypertension in 34 and vasospasm in one case. The most common systemic cause was hypocarbia, defined as a $PaCO_2$ < 3·7 kPa (28 mmHg) ($n = 21$), followed by systemic hypotension, defined as systolic blood pressure < 90 mmHg ($n = 8$), arterial hypoxia, defined as an SaO_2 < 90% ($n = 6$), and anaemia ($n = 1$). Combinations of both systemic and cerebral causes were observed in six episodes.

UNIQUE INFORMATION PROVIDED BY MONITORING OF $SjvO_2$

An example of hypocarbia producing jugular venous desaturation is given in figure 1 which illustrates two patients with very different responses to lowering PCO_2 to 3·3 kPa (25 mmHg). One patient developed a very low $SjvO_2$ and a low CBF which returned to
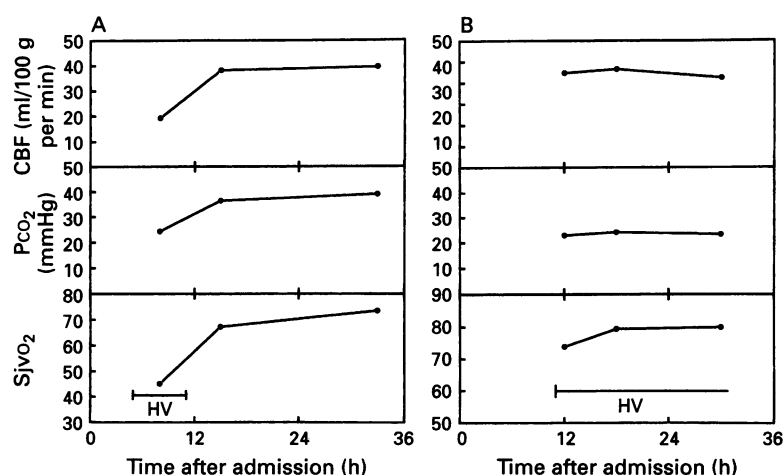

Figure 1   Two patients exhibiting different effects of hyperventilation on $SjvO_2$. The patient whose data are shown in A had a marked decrease in CBF and $SjvO_2$ when (PCO_2 was decreased to 3·3 kPa (25 mmHg). These abnormalities reversed when the ventilator was adjusted to normalise PCO_2. The patient whose data is shown in B had acceptable CBF and $SjvO_2$ with similar PCO_2 values of 2·7–3·3 kPa (20–25 mmHg).

Table 1   Demographic and injury related parameters which were examined for an effect on the relationship between desaturations and outcome

	Number of desaturations				Three month Glasgow outcome score			
	None	One	Multiple	p value*	GR/MD	SD/V	Dead	p value*
Number	70	27	19		40	40	36	
Age (years)	31·3 (14·9)	28·5 (11·6)	36·2 (16·9)	0·23	28·8 (13·3)	31·2 (14·0)	35·0 (16·5)	0·18
Sex (% male)	65 (92·9)	23 (85·2)	16 (84·2)	0·37	37 (92·5)	35 (87·5)	32 (88·9)	0·75
Severity of injury								
ER GCS (score)	7·2 (3·0)	6·3 (2·5)	7·0 (3·5)	0·45	7·6 (3·1)	6·5 (2·7)	6·6 (3·2)	0·21
ER pupils (% normal)	53 (75·7)	16 (59·3)	14 (73·7)	0·25	34 (85·0)	29 (72·5)	20 (55·6)	0·004
Day 1 GCS (score)	7·0 (2·1)	6·6 (2·2)	6·4 (3·2)	0·61	8·1 (2·3)	6·7 (1·8)	5·5 (2·2)	<0·001
Day 1 pupils (% normal)	56 (81·2)	24 (88·9)	15 (78·9)	0·62	39 (97·5)	31 (79·5)	25 (69·4)	<0·001
Injury type (%)				0·87				0·001
Closed head injury								
Diffuse	23 (60·5)	10 (26·3)	5 (13·20)		21 (55·3)	11 (28·9)	6 (15·8)	
Hematoma	36 (58·1)	14 (22·6)	12 (19·4)		16 (25·8)	19 (30·6)	27(43·5)	
Gunshot wounds	11 (68·8)	3 (18·8)	2 (12·5)		3 (18·8)	10 (62·6)	3 (18·8)	
Presence of systemic injuries								
ISS (score)	26 (6)	26 (4)	27 (5)	0·88	26 (7)	26 (5)	27(4)	0·95

*p value is for analysis of variance when mean values were compared and the χ squared test used with categorical data. Values in parentheses are standard deviations.
ER = emergency room (post resuscitation); GCS = Glasgow coma score; ISS = injury severity score; GR = good recovery; MD = moderate disability; SD = severe disability; V = vegetative.
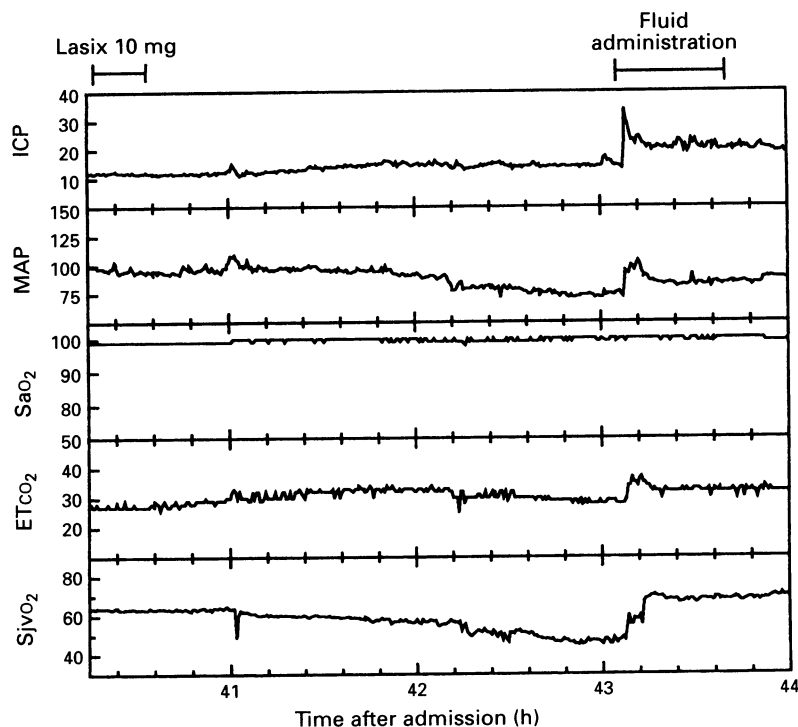
Figure 2   *SjvO₂ recording from a patient who developed jugular venous desaturation as a result of systemic hypotension after a small dose of frusemide (furosemide) had been given. As the blood pressure gradually decreased from 100 to 75 mmHg, the SjvO₂ also decreased. Both the MAP and the SjvO₂ normalised when intravenous fluid was given. ETCO₂ = end tidal CO₂.*

75 mmHg after a dose of frusemide (furosemide) was given. Although the CPP remained >60 mmHg, the $SjvO_2$ dropped to below 50%. The MAP and $SjvO_2$ values normalised with fluid administration.

RELATIONSHIP OF JUGULAR VENOUS DESATURATION AND CEREBRAL BLOOD FLOW

Twenty (17%) of the patients had a reduced CBF, averaging 0·352 (SD 0·083) ml/g per minute; 65 (56%) had an elevated and 31 (27%) a normal CBF, averaging 0·606 (SD 0·124) ml/g per minute and 0·422 (SD 0·90) ml/g per minute, respectively. As shown in figure 3, patients with a reduced CBF were twice as likely to have episodes of jugular venous desaturation as patients with a normal or elevated CBF. Fourteen (70%) of the 20 patients in the reduced CBF group had either single or multiple episodes of desaturation, compared with nine (29%) in the normal CBF group, and 22 (34%) in the elevated CBF group.

RELATIONSHIP OF JUGULAR VENOUS DESATURATION AND NEUROLOGICAL OUTCOME

Forty (34·5%) of the patients had a favourable outcome three months after injury (20 = good recovery, 20 = moderate disability); 40 (34·5%) survived with a severe disability ($n = 26$) or were vegetative ($n = 14$), and 36 (31%) died. The incidence of jugular venous desaturation was strongly associated with neurological outcome (fig 4). The mortality rate of patients with no episodes of desaturation was 17%, compared to 41% in patients with one desaturation, and 68% in those with multiple desaturations. The percentage of patients with a poor neurological outcome (GOS of severe disability, vegetative, or dead) was 55% in patients with no desaturation, 74% in those with one desaturation, and 89·5% in those with multiple desaturations.

The total duration of time that $SjvO_2$ was less than 50% was significantly longer (p = 0·002) in the patients who died than in the patients who survived. The total duration of all the episodes of desaturation averaged

normal after normalising PCO_2. The other patient had acceptable $SjvO_2$ and CBF values at a PCO_2 of 3·3 kPa (25 mmHg). Monitoring of $SjvO_2$ allowed identification of those patients who might have been adversely affected by hyperventilation. Although not all patients were hyperventilated in the present series, 21 episodes of hypocapnia-induced desaturation were identified in 19 of the 116 patients.

Although conventional teaching has been that a CPP of 50 mmHg is normal, recent studies have suggested that a CPP level of 70 or even 80 mmHg may be desirable in head injured patients.[14][15][17][18] In addition, the optimal CPP may vary from patient to patient. Figure 2 illustrates a patient who developed desaturation when his MAP fell from 95 to
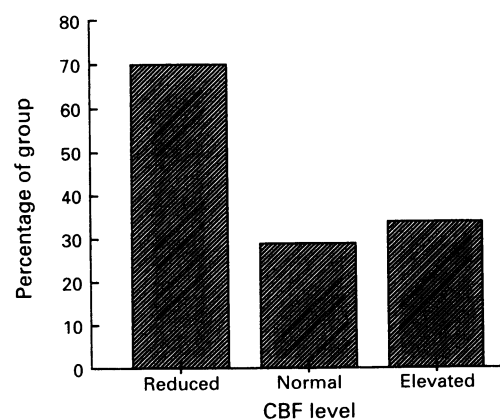

Figure 3   *Patients with a reduced CBF had twice the incidence of one or multiple episodes of jugular desaturation, compared to patients with a normal or elevated CBF.*
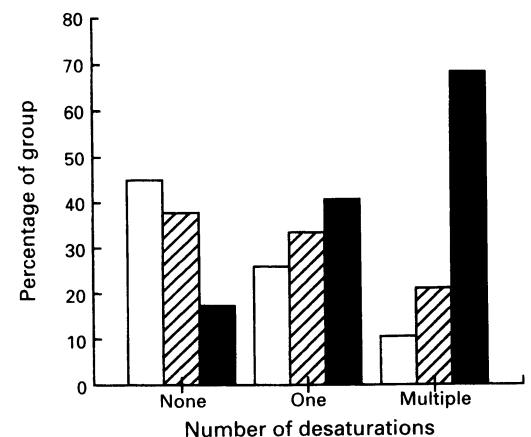

Figure 4   *Occurrence of jugular venous desaturation was strongly associated with a high mortality rate and a poor outcome; 3 month Glasgow outcome scale: □ good recovery/moderate disability; ▨ severe disability/vegetative; ■ dead.*

Table 2 Systemic physiological variables that were examined for an effect on the relationship between desaturations and outcome

	Number of desaturations				Three month Glasgow outcome score			
	None	One	Multiple	p value*	GR/MD	SD/V	Dead	p value*
Number of patients	70	27	19		40	40	36	
BP (mmHg)								
Mean	93 (7)	91 (10)	85 (11)	0·005	94 (7)	94 (7)	85 (11)	<0·001
Lowest	68 (9)	63 (14)	60 (13)	0·010	70 (8)	69 (10)	57 (12)	<0·001
PaO₂								
(mean) (mmHg)	173 (39)	184 (46)	160 (39)	0·138	174 (44)	170 (36)	177(45)	0·748
PaCO₂								
(mean) (mmHg)	32 (4)	30 (3)	31 (3)	0·046	32 (4)	32 (4)	29 (2)	0·001
Haemoglobin								
(mean) (g/dl)	10·8 (2·1)	10·1 (1·8)	10·6 (1·8)	0·390	10·6 (2·0)	10·7 (2·0)	10·4 (1·9)	0·802
Temperature								
Mean (°C)	100·5 (6)	100·2 (0·7)	100·1 (1·0)	0·068	100·4 (0·6)	100·6 (0·5)	100·0 (0·9)	<0·001
Highest	103·0 (0·1)	102·9 (0·2)	102·7 (0·3)	0·613	102·7 (0·1)	103·1 (0·2)	102·9 (0·2)	0·166

*p value is for analysis of variance. Values in parentheses are standard deviations. BP = mean blood pressure; PaO_2 = arterial PO_2; $PaCO_2$ = arterial PCO_2.

1·3 (SD 1·6) hours in the patients who died, compared to 0·4 (SD 0·8) hours in patients with a good recovery or moderate disability, and 0·4 (SD 1·1) hours in patients who had a severe disability or were vegetative.

RELATIONSHIP OF OTHER FACTORS TO NEUROLOGICAL OUTCOME

Many factors have been associated with outcome after severe head injury, including age, severity of injury, type of injury, presence of systemic injuries, severity of intracranial hypertension, and level of CBF. The relationship of the demographic factors to neurological outcome in the 116 patients are summarised in table 1. Age was not significantly associated with outcome, although there was a trend for the patients with a favourable outcome to be younger.

The severity of injury was examined in the emergency room by using the best GCS and pupillary reactivity on day 1 after injury. The GCS after resuscitation in the emergency room was not significantly associated with neurological outcome, but the best GCS on day 1 after injury had a strong relationship to outcome, averaging 8·1 (SD 2·3) in patients with a favourable outcome, compared to 6·5 (SD 2·7) in patients with a poor outcome, and 5·5 (SD 2·2) in patients who died. Pupillary

size and reactivity in the emergency room and on day 1 were strongly related to outcome. Patients who died of their head injury had a higher incidence of one or both pupils being dilated and unreactive.

Sixteen (14%) of the patients had a gunshot wound to the head and 100 (86%) had closed head injuries. The type of closed head injury was classified as described by the Traumatic Coma Data Bank group.[19] Thirty-eight (38%) of the patients with closed head injuries had one of the four categories of diffuse injury (6-I, 19-II, 11-III, and 2-IV). Fifty-four (54%) had evacuated haematomas of at least 25 ml in volume, and eight (8%) had unevacuated haematomas. The neurological outcome of patients with diffuse lesions was significantly better than for those with haematomas and gunshot wounds. Twenty-one (55%) of the 38 patients with diffuse lesions had a favourable outcome compared to only 16 (26%) of the 62 patients with haematomas and three (19%) of the 16 patients with gunshot wounds. The mortality rate was 16% in patients with diffuse injuries, 43·5% in those with haematomas, and 19% in those with gunshot wounds.

The injury severity score (ISS) was used to assess whether multiple systemic injuries were associated with neurological outcome. An ISS

Table 3 Cerebral physiological variables that were examined for an effect on the relationship between desaturations and outcome

	Number of desaturations				Three month Glasgow outcome score			
	None	One	Multiple	p value*	GR/MD	SD/V	Dead	p value*
Number of patients	70	27	19		40	40	36	
ICP (mmHg)								
Mean	17 (6)	22 (9)	21 (12)	0·008	15 (5)	16 (5)	25 (10)	<0·001
Highest	35 (12)	47 (22)	43 (24)	0·009	33 (11)	34 (11)	53 (23)	<0·001
CPP (mmHg)								
Mean	76 (9)	70 (17)	64 (20)	0·003	78 (7)	78 (9)	60 (17)	<0·001
Lowest	48 (14)	33 (28)	33 (30)	0·001	53 (10)	49 (15)	22 (26)	<0·001
CBF (ml/g per min)								
Mean	0·523 (0·146)	0·516 (0·147)	0·464 (0·178)	0·314	0·544 (0·163)	0·530 (0·148)	0·456 (0·130)	0·024
Lowest	0·459 (0·156)	0·415 (0·140)	0·429 (0·186)	0·421	0·465 (0·163)	0·433 (0·155)	0·434 (0·157)	0·590
SjvO₂ (%)								
Mean	71 (6)	69 (7)	68 (5)	0·059	69 (5)	70 (5)	71 (7)	0·325
Lowest	50	43(1)	34 (2)	<0·001	51 (7)	50 (9)	45 (13)	0·027
No of times <50%	0	1	>1		0·3 (0·6)	0·4 (0·7)	1·0 (0·8)	<0·001
Time <50% (h)	0	1·4 (0·3)	1·9 (0·4)	<0·001	0·4 (1·1)	0·4 (0·8)	1·3 (1·6)	0·002
CMRO₂ (μmol/g per min)								
Mean	1·00 (0·39)	1·09 (0·53)	0·94 (0·30)	0·433	1·12 (0·41)	1·03 (0·43)	0·86 (0·35)	0·017
Lowest	0·62 (0·28)	0·69 (0·47)	0·54 (0·29)	0·340	0·73 (0·35)	0·61 (0·32)	0·52(0·30)	0·017
CVR (mmHg/ml per 100 g per min)								
Mean	1·94 (0·58)	1·57 (0·50)	1·79 (0·75)	0·480	1·65 (0·49)	1·73 (0·67)	1·56 (0·61)	0·491

*p value is for analysis of variance. Values in parentheses are standard deviations. ICP = intracranial pressure; CPP = cerebral perfusion pressure; CBF = cerebral blood flow; $SjvO_2$ = jugular venous oxygen saturation; $CMRO_2$ = cerebral metabolic rate of oxygen; CVR = cerebral vascular resistance.

Table 4 Final logistic regression model for poor neurological outcome (severe disability, vegetative, or dead three months after injury) as dependent variable

Variable/Category	Odds ratio	p value
Number of desaturations		0·0348 (overall variable)
Multiple	14·46	0·012
One	2·09	0·289
None	1·00	
Type of injury		0·0652 (overall variable)
Gunshot wound	4·39	0·253
Closed head injury— hematoma	4·60	0·021
Closed head injury— diffuse	1·00	
Day 1		
Glasgow coma score		0·0025 (overall variable)
3–5	41·79	0·000
6–8	8·61	0·005
9–15	1·00	
Pupillary reactivity		
One or both dilated and unreactive	21·55	0·052
Neither unreactive	1·00	
Highest temperature (per °F increase)	2·23	0·023
Lowest cerebral perfusion pressure (per mmHg increase)	0·94	0·004
Age (per year increase)	1·06	0·017

of 25 indicates the presence of only a severe head injury. The presence of systemic injuries increases the score, depending on their severity. The ISS was not significantly related to neurological outcome.

The relationships of the physiological variables to outcome are listed in tables 2 and 3. The mean and the highest recorded ICP values, the mean and the lowest blood pressure values, the mean and the lowest CPP values, the mean $PaCO_2$, and the mean temperature were all significantly related to outcome. The mean CBF was lower in patients who died. The mean and the lowest cerebral metabolic rate of oxygen ($CMRO_2$) values were significantly lower in the patients who died.

Logistic regression analysis

To determine whether the difference in neurological outcome with jugular desaturation reflected the effect of other co-variates, logistic regression analysis was used as described above. The final best fit model is shown in table 4. When the effect of jugular desaturation was adjusted for all other co-variates which were found to be significant, including age, severity of injury (day 1 GCS and pupillary reactivity), type of injury (CT category), lowest recorded CPP, and highest recorded temperature, the incidence of jugular desaturation was still significant (p = 0·03). A single episode of desaturation resulted in a doubling of the probability of a poor outcome, and multiple episodes of desaturation were associated with a 14-fold higher probability of a poor outcome.

Discussion

Although the critical level for $SjvO_2$, below which the brain is ischaemic, has not been established definitely, a few studies suggest that an $SjvO_2$ value of <50% can be related to cerebral ischaemia. Continuous monitoring of oxygen saturation in the lateral sinus, during carotid endarterectomy in 50 patients, revealed that cerebral dysfunction was found in patients with a venous oxygen saturation (SvO_2) <50%, and there were no cases of dysfunction when SvO_2 remained at least 60%.[20] EEG slowing appeared when jugular venous $PjvO_2$) fell to <2·5 kPa (19 mmHg) (an $SjvO_2$ value of about 40%) in 19 healthy volunteers.[21] Neurological deterioration occurred in head injured patients when the $SjvO_2$ value was <30% for more than 10 minutes.[22]

The critical duration of ischaemia necessary to produce injury is not known, but experimental studies suggest that brief episodes of ischaemia can be injurious, particularly if they are repeated. Cerebral oedema and brain tissue injury were more severe in gerbils sacrificed 24 hours after three separate five-minute periods of bilateral carotid occlusion at one-hour intervals than in animals which received a single 15-minute occlusion.[23–25]

There are several limitations to the methodology of the present study which might have underestimated the actual incidence of cerebral ischaemia. $SjvO_2$ values are a measure of global cerebral oxygenation, and significant regional ischaemia can occur in the presence of a normal $SjvO_2$ value. Systemic insults on admission to the hospital have been shown to increase mortality rate approximately twofold.[3 4 26] Andrews et al[27] found a significant incidence of secondary insults during within hospital transfers of head injured patients. Insults before admission and during transfer were not monitored in the present study. Very low CBF values, suggesting ischaemia have been described after a severe head injury, but only commonly within the first eight hours.[28 29] The average start time of $SjvO_2$ monitoring in the present study was 17 hours after admission.

The present study demonstrates that episodes of jugular venous desaturation are common during the first five days after severe head injury, and that these episodes are the result of both systemic and cerebral causes, some of which are treatable. The occurrence of jugular venous desaturation is strongly associated with a poor neurological outcome. Although a cause and effect relationship with outcome cannot be established in the present study, it is reasonable to hypothesise that monitoring $SjvO_2$ might allow early identification and therefore treatment of a number of causes of secondary injury to the brain.

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